Available online at www.sciencedirect.com
ScienceDirect
Cognitive and Behavioral Practice 17 (2010) 88–101
Cognitive and Behavioral Practice
www.elsevier.com/locate/cabp

Unified Protocol for the Transdiagnostic Treatment of Emotional Disorders: Protocol Development and Initial Outcome Data

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The Unified Protocol (UP) is a transdiagnostic, emotion-focused cognitive-behavioral treatment developed to be applicable across the emotional disorders. The UP consists of 4 core modules: increasing emotional awareness, facilitating flexibility in appraisals, identifying and preventing behavioral and emotional avoidance, and situational and interoceptive exposure to emotion cues. Here we present data from 2 open clinical trials. In the first trial, an initial version of the UP was administered to a heterogeneous clinical sample, yielding significant pre- to posttreatment effects across disorders on a variety of measures. Analyses of clinical significance demonstrated modest results, with 56% of participants achieving responder status and 33% achieving high end-state functioning. Further manual development ensued, resulting in specific modifications and enhancements to core treatment components, and a second trial presents data from an additional pilot study of this revised version of the UP. Results from this trial demonstrated more robust treatment effects, with 73% achieving responder status and 60% achieving high end-state functioning. Results improved further at 6-month follow-up, with 85% classified as treatment responders and 69% achieving high end-state functioning. Implications for the treatment of emotional disorders as well as dimensional conceptualizations of psychopathology are discussed.

Anxiety and mood disorders disrupt the lives of millions of Americans each year, with lifetime prevalence rates for anxiety disorders estimated at 29% of the population, and mood disorders at 21% (Kessler, Berglund, Demler, Jin, & Walters, 2005). Anxiety disorders alone represent a cost to this country of over $42 billion annually (Greenberg et al., 1999). In addition, anxiety disorders are associated with high rates of comorbidity, with 12-month rates of comorbid anxiety and/or depression estimated as high as 40% to 80% (Kessler, Chiu, Demler, & Walters, 2005). Clearly, effective treatments for anxiety and mood disorders that can be widely disseminated are sorely needed to address this significant public health risk. In service of this goal, a number of evidence-based cognitive-behavioral treatments targeting specific anxiety and mood disorders have been developed over the last 20-plus years (Antony & Stein, 2009; Barlow, 2002; Norton & Price, 2007; Smits & Hoffman, 2008). However, along with the development of these effective treatments has come a proliferation of diagnosis-specific treatment manuals, placing a significant burden on practicing clinicians who wish to deliver empirically supported treatments to their patients, and hampering efforts at widespread dissemination of evidence-based psychological treatments.

Recent scientific advances suggest that there may be more that unites anxiety and mood disorders than previously conceived, potentially making the need for numerous diagnosis-specific treatments obsolete and opening the possibility for a more parsimonious application of evidence-based treatments in clinical practice. Over the last several years, research emerging from the fields of neuroscience, emotion science, and descriptive and functional psychopathology has begun to identify common, higher-order factors that underlie anxiety, mood and related emotional disorders. For example, using structural equation modeling, Brown, Chorpita, and Barlow (1998) found that the covariance among latent factors corresponding to a range of emotional disorders including unipolar depression, social anxiety (SAD), generalized anxiety disorder (GAD), obsessive-compulsive disorder (OCD), and panic disorder with agoraphobia (PDA) was explained by the higher-order factors of negative and (low) positive affect. Specifically, negative affect loaded positively on all five *DSM-IV* disorder categories (Brown et al., 1998). Consistent with this structural model, preliminary investigations emerging from the field of affective neuroscience consistently demonstrate similar increased activation in key neural structures implicated in the generation of negative affect in individuals with anxiety and mood disorders relative to

1077-7229/09/88–101$1.00/0

healthy controls (for a review, see Etkin & Wager, 2007). This pattern has been demonstrated in SAD (Lorberbaum et al., 2004; Phan, Fitzgerald, Nathan, & Tancer, 2006; Tillfors, Furmark, Marteinsdottir, & Fredrikson, 2002) posttraumatic stress disorder (PTSD) (Shin et al., 2005; Williams et al., 2006), GAD (Hoehn-Saric, Schlund, & Wong, 2004; McClure et al., 2007), specific phobia (Paquette et al., 2003; Straube, Glauer, Dilger, Mentzel, & Miltner, 2006), and depression (Goldapple et al., 2004; Mayberg et al., 1999; Siegle, Thompson, Carter, Steinhauer, & Thase, 2007). Converging evidence for the specific contribution of negative affect across disorders also comes from recent behavioral investigations of emotion regulation and emotional processing. These studies have increasingly found individuals suffering from anxiety and mood disorders to endorse more frequent and intense experiences of negative affect than healthy individuals (Campbell-Sills, Barlow, Brown, & Hofman, 2006; Mennin, Heimberg, & Turk, 2005) and view these experiences as more aversive (Roemer, Salters, Raffa, & Orsillo, 2005).

Other investigations into unifying features across mood and anxiety disorders have identified common cognitive, behavioral, and emotion-regulation processes that may serve as targets for therapeutic change. For example, the association between processing biases found across anxiety and mood disorders and negative emotional experiences has been long established (Beck & Clark, 1997; Dalgleish & Watts, 1990; Matthews & MacLeod, 2002; McLaughlin, Borkovec, & Sibrava, 2007; Mobini & Grant, 2007; Watkins, Moberly, & Moulds, 2008). Behavioral avoidance arising out of attempts to manage increased negative affect, potentially emerging from these cognitive biases, has been proposed as a key pathological feature common across anxiety and mood disorders (Brown & Barlow, 2009). Finally, the growing literature on emotion regulation has found deficits in the ability to regulate emotional experiences, emerging out of unsuccessful efforts to avoid or dampen the intensity of negative emotions, to be prevalent across anxiety and mood disorders (e.g., Cambpell-Sills et al., 2006; Liverant, Brown, Barlow, & Roemer, 2008; Mennin et al., 2005; Tull, 2006).

Extant single-diagnosis treatments for anxiety disorders also share common features, such as cognitive restructuring, prevention of avoidance, and exposure-based procedures (Barlow, Allen, & Choate, 2004). In support of a focus on higher-order transdiagnostic features, Brown (2007) found that the temporal covariance of the *DSM-IV* disorder constructs was fully accounted for by change in negative affect over a 2-year interval. This suggests that negative affect represents a unifying construct accounting for the covariance of emotional disorders. Thus, addressing the core affective processes contributing to an increase in negative affect present across the emotional disorders, rather than discrete, disorder-specific heterogeneous symptoms, may more efficiently target the root of these disorders and result in reductions in co-occurring disorder symptoms.

In addition to our own efforts (Barlow et al., 2004), Norton and others have proposed a somewhat similar set of transdiagnostic therapeutic principles for the anxiety disorders (Erickson, Janeck, & Tallman, 2007; McEvoy & Nathan, 2007; Norton & Hope, 2005; Norton & Philipp, 2008), although to date these have been delivered primarily in group format. Fairburn et al. (2009) and Fairburn, Cooper, and Shafran (2003) have developed a transdiagnostic protocol for eating disorders to address the particularly large number of patients who meet "not otherwise specified" (NOS) criteria. In addition, some investigators have begun to consider the totality of extant evidence-based therapeutic principles and how they could be integrated in various ways to address the full range of psychopathology in a transdiagnostic manner (Harvey, Watkins, Mansell, & Shafran, 2004). As Mansell, Harvey, Watkins, and Shafran (2009) point out, the scientific principles of parsimony and pragmatism strongly support a transdiagnostic approach if it is feasible.

Development of the Unified Transdiagnostic Treatment for Emotional Disorders

In response to these advances, we developed the Unified Protocol for the Treatment of Emotional Disorders (UP), a transdiagnostic, emotion-focused cognitive-behavioral treatment (CBT) (Barlow, Boisseau, Ellard, Fairholme, & Farchione, 2008). The UP was developed to be applicable across anxiety and mood disorders, as well as other disorders in which anxiety and emotion dysregulation play a significant role, such as many somatoform and dissociative disorders. The focus in the UP on common underlying factors reflects scientific advances leading to more dimensional conceptions of psychopathology, and represents a movement away from the extreme diagnostic splitting evident in *DSM-IV* that has resulted in the proliferation of diagnosis-specific treatments. Further, this approach renders moot the issues of comorbidity, NOS diagnoses, and subthreshold presentations among anxiety and mood disorders allowing for more focused and simplified treatment planning.

The UP has emerged out of decades of research leading to the development of effective cognitive and behavioral treatments for anxiety and mood disorders (Barlow, 2002). The development of the UP began with the distillation of key principles from traditional empirically supported CBT treatments (e.g., Barlow, 1985; Barlow & Cerny, 1988; Barlow & Craske, 1989; Beck, 1972; Beck, Rush, Shaw, & Emery, 1987; Craske, Barlow, & O'Leary, 1992) and advances in research on adaptive

emotion regulation (e.g., Campbell-Sills et al., 2006; Gross, 1998; Mennin et al., 2005). Thus, at the core of the UP are the fundamental principles of traditional CBT, including emphases on extinction learning through preventing avoidance, behavioral exposure and the identification and modification of maladaptive cognitions. However, the focus of extinction training now extends to anxiety focused on interoceptive cues, including those associated with intense emotions, an extension of a concept first utilized in panic disorder (Barlow, 1988; Barlow, Craske, Cerny, & Klosko, 1989; Craske, 1991). The UP also expands upon traditional CBT by more explicitly focusing on the interaction of thoughts, feelings, and behaviors in generating internal emotional experiences, and the subsequent role of emotion (dys) regulation in modifying these experiences. As such, the UP emphasizes the adaptive, functional nature of emotions, helps facilitate greater tolerance of emotions, and seeks to identify and correct maladaptive attempts to regulate emotional experiences. The initial version of the UP treatment manual included sessions targeting antecedent cognitive reappraisal emphasizing two core thinking traps: overestimating the probability of negative events occurring (jumping to conclusions) and catastrophizing (thinking the worst) (Craske & Barlow, 1989); the prevention of emotional avoidance and increased emotional awareness; and the identification and modification of emotion-driven action tendencies (Barlow, 1988; termed "emotion driven behaviors," or "EDBs"). Treatment concepts were tied together in the final phase of treatment through engagement in interoceptive and situationally based emotion exposures, emphasizing the elicitation of and exposure to both situational and internal emotional experiences. For a more detailed description of the initial version of the UP, see Allen, McHugh, and Barlow (2008).

This first, early version of the UP was pilot-tested in a diagnostically heterogeneous sample of 18 patients presenting for treatment at the Center for Anxiety and Related Disorders at Boston University (CARD; see Study 1 below). Initial pilot-testing allowed us to acquire valuable clinical insight into how well patients acquired and adopted the core skills of the treatment, as well as how treatment concepts could be presented in a more logical progression. This in turn led us to consider ways in which the treatment could be improved upon further. Hence, initial pilot-testing was followed by revision of the treatment manual with the aim of enhancing patient learning and acquisition of core emotion-regulation skills, thereby facilitating the extinction of both internally and situationally cued anxiety. The revised version of the protocol was subsequently pilot-tested in an additional heterogeneous sample of 15 patients (Study 2). Here, we present data from these two open trials of the UP.

Study 1: Pilot-Test of Initial Version of the UP

The initial version of the UP was pilot-tested in a sample of patients whose principal diagnoses spanned the anxiety disorders, including GAD, OCD, SAD, PTSD, and PDA, as well as major depressive disorder (MDD) and dysthymia. Consistent with epidemiological accounts (Kessler et al., 2005), and prior research (Brown, Campbell, Lehman, Grisham, & Mancill, 2001), the sample evidenced high rates of comorbidity (see below). We hypothesized that treatment using the UP would result in reductions in clinical disorder severity across these disorder categories, as well as improvement in comorbid symptoms. We also hypothesized that treatment with the UP would result in improvement across the anxiety disorders on general measures of depression and anxiety, lower endorsement of negative affect, and reductions in symptom interference in daily functioning.

Method

Participants

Participants were recruited from a pool of individuals seeking treatment at Boston University's CARD. All individuals were assessed using the Anxiety Disorders Interview Schedule for DSM-IV–Lifetime Version (ADIS-IV-L; DiNardo, Brown, & Barlow, 1994) and were contacted for participation if they received a principal diagnosis of an anxiety disorder (see below for a description). Individuals were excluded only if treatment for an anxiety disorder might not be the primary clinical priority; for instance, current significant suicidal ideation, current substance dependence diagnosis, or a history of mania or a psychotic disorder.

Twenty-four patients consented to treatment. Two of the 24 who had consented dropped out of treatment. Of the remaining 22 patients, 2 did not complete posttreatment assessments, and 2 had incomplete posttreatment assessments. Therefore, complete posttreatment data were available for 18 participants and are included in the present study. Participants were 58.8% female (n=11). The mean age was 30 years (SD=10.64) and participants ranged from 18 to 54 years old. The sample was primarily Caucasian (n=17). Nine individuals were taking psychotropic medications at the time of enrollment and randomization. All individuals were stable on the same dose for at least 3 months prior to enrolling in the study as a condition for participation in the study, and all agreed to maintain these dosages and medications for the duration of the study. Sixteen individuals had received prior psychosocial treatment for anxiety or mood disorders. Principal diagnoses represented by the sample included: GAD (n=3), SAD (n=4), OCD (n=3), PDA (n=4), PTSD (n=1), MDD (n=2), and hypochondriasis (n=1). Two individuals had co-principal diagnoses (a

diagnosis of equal severity). For these individuals the co-principal diagnoses were anxiety not otherwise specified and hypochondriasis (n=1), and MDD and SAD (n=1). Participants in Study 1 had an average number of 1.94 diagnoses at pretreatment (SD=0.64; range 1 to 3 diagnoses). Additional or comorbid diagnoses included: GAD (n=4), SAD (n=4), OCD (n=2), MDD (n=3), dysthymia (n=2), specific phobia (n=1), and impulse control NOS (n=1).

Measures

The assessment of treatment effects in a heterogenous sample poses a unique challenge. Therefore, in order to adequately assess changes in symptoms and functioning across multiple anxiety and mood disorders simultaneously, an extensive assessment battery was administered to participants. These assessments allowed us to ascertain the effects of treatment on three broad areas, including changes in clinical diagnoses and diagnostic symptom severity using a clinician-rated diagnostic assessment; changes in both general and diagnosis-specific symptoms assessed through self-report measures; and changes in the level of symptom interference in daily functioning, also assessed by self-report. Descriptions of included measures are detailed below.

Anxiety Disorders Interview Schedule for DSM-IV–Lifetime Version (ADIS-IV-L; DiNardo et al., 1994). This semistructured, diagnostic clinical interview focuses on *DSM-IV* diagnoses of anxiety, mood, somatoform, and substance use disorders. The information derived from the interview using the ADIS allows clinicians to determine differential diagnoses and gain a clear understanding of the level of impairment and severity of each diagnosis. Principal and additional diagnoses are assigned a clinical severity rating (CSR) on a scale from 0 (*no symptoms*) to 8 (*severely disturbing/disabling*), with a rating of 4 or above (*definitely disturbing/disabling*) passing the clinical threshold for *DSM-IV* diagnostic criteria. In instances where the patient meets criteria for two or more current diagnoses, the principal diagnosis is assigned as the diagnosis with the higher CSR, representing the greatest amount of interference and/or distress, and the remaining diagnoses become additional (comorbid) diagnoses. Occasionally, co-principal diagnoses are assigned when diagnoses are determined to be equally severe and interfering. This measure has demonstrated acceptable to excellent interrater reliability for the anxiety and mood disorders (Brown, DiNardo, Lehman, & Campbell, 2001).

The ADIS-IV-L was administered during the first assessment, and an abbreviated version assessing only current diagnoses was administered at posttreatment assessments. Posttreatment assessments were administered by independent evaluators (IEs) naïve to previous assessment results. All IEs were doctoral students at the CARD who had undergone extensive training on the administration and scoring of the ADIS-IV-L (see Brown et al., 2001).

Beck Depression Inventory-II (BDI-II; Beck, Steer, & Brown, 1996). The BDI-II is perhaps the most widely used self-report measure to assess current depressive symptoms, and was included as a general measure of depressive symptoms across the disorders. It contains 21 items focusing on the levels of depressive symptoms over the past 2 weeks. Participants are asked to circle the number next to the statement that best corresponds to how they felt over the past week. Scores range from 0 to 63, with higher scores indicating greater depressive symptoms.

Beck Anxiety Inventory (BAI; Beck & Steer, 1990; Steer, Ranieri, Beck, & Clark, 1993). The BAI was included as a general measure of anxiety-related symptoms across the disorders. The BAI also contains 21 items scored in a similar way and focuses on common symptoms that are more unique to anxiety, such as somatic and certain cognitive symptoms.

Positive and Negative Affect Scale (PANAS; Watson, Clark, & Tellegen, 1988). The PANAS was included to assess levels of positive and negative affect across the disorders. The PANAS is a brief, reliable, and valid self-report measure of positive and negative affect. It consists of 20 feeling or emotion words. Respondents rate each emotion word on a scale ranging from 1 (*very slightly or not at all*) to 5 (*extremely*), indicating the extent to which they experienced that emotion or feeling during the past few weeks. The PANAS allows for the assessment of core negative affect as well as deficits in positive affect. The PANAS has shown excellent convergent and divergent validity.

Obsessive-Compulsive Inventory–Revised version (OCI-R; Foa, Kozak, Salkovskis, Coles & Amir, 1998). The OCI-R was included to assess symptoms related to OCD. The OCI-R is an 18-item self-report measure designed to measure distress related to obsessive and compulsive symptoms. Respondents are asked to rate the extent to which they were distressed or bothered by particular symptoms using a scale of 1 (*not at all*) to 4 (*extremely*). This revised version of the original 42 item scale removes ratings of frequency and eliminates overlap among subscales. It has demonstrated good to excellent internal consistency, test-retest reliability, and convergent validity (Foa et al., 1998).

Panic Disorder Severity Scale (PDSS-SR; Houck, Spiegel, Shear, & Rucci, 2002). The PDSS-SR was included as a measure of symptoms related to panic disorder. The PDSS-SR is a self-report version of the clinician-administered PDSS (Shear et al., 1992). It is designed to assess panic attack frequency, distress during panic attacks, severity of anticipatory anxiety, fear and avoidance of agoraphobic

situations, fear and avoidance of panic-related sensations, impairment in work functioning, and impairment in social functioning. The self-report version was modified to provide questions respondents can answer independently and to change the timeframe from "past month" to "past week" to prevent recall bias. The PDSS-SR was found to be highly correlated with the interview version, demonstrated good internal consistency and test-retest reliability, and was found to be sensitive to change (Houck et al., 2002).

Penn State Worry Questionnaire (PSWQ; Meyer, Miller, Metzger, & Borkovec, 1990). The PSWQ was included to assess symptoms related to GAD. The PSWQ is a 16-item self-report questionnaire designed to assess the tendency to worry as well as intensity and excessiveness of worry. Respondents indicate the extent to which items represent tendencies typical to them on a scale of 1 (*not at all typical of me*) to 5 (*very typical of me*). The PSWQ has demonstrated good internal consistency and test-retest reliability (Molina & Borkovec, 1994).

Social Interaction Anxiety Inventory (SIAS; Mattick & Clarke, 1998). The SIAS was included to assess symptoms related to social phobia. The SIAS is 20-item self-report measure developed to assess cognitive, affective, and behavioral reactions to social interactions. Items are rated on a 5-point scale ranging from 0 (*not at all characteristic or true of me*) to 4 (*extremely characteristic of me*). The SIAS has demonstrated high internal consistency and test-retest reliability, and has been shown to be sensitive to treatment (e.g., Cox et al., 1998).

Work and Social Adjustment Scale (WSAS; Marks, 1986). The WSAS is a 5-item measure asking participants to rate the degree of interference caused by their symptoms in work, home management, private leisure, social leisure, and family relationships. Interference is rated over the past week on a 0 to 8 scale (0 = *not at all* interfering to 8 = *severe interference*). The WSAS is a descriptive measure of subjective interference in various domains of living, and has been successfully used in previous studies (e.g., Brown & Barlow, 1995).

Treatment

A maximum of fifteen 60-minute individual treatment sessions were allowed in Study 1. Patients who completed a full course of treatment were seen on average 13 total sessions (range 8–15) of 15 total allowable sessions. Treatment was comprised of four main components: (1) psychoeducation about emotions, including a review of the functional nature of emotions; (2) alteration of antecedent cognitive misappraisals; (3) prevention of emotional avoidance; and (4) modification of emotion-driven behaviors (EDBs). Treatment emphasized emotion exposures (provoking emotion expression) through situational, internal, and somatic (interoceptive) cues, as well as standard mood inductions. For a more complete description of treatment, see Allen et al., (2008).

Therapists and Treatment Integrity

Therapists for the study were six doctoral students with 1 to 4 years of experience, providing treatment under the close supervision of a licensed senior team member. Treatment adherence was monitored during weekly supervision and manual development meetings.

Results

Efficacy at Posttreatment Assessment

Descriptive statistics and effect size estimates for the primary study variables are shown in Table 1. In order to assess the impact of treatment on diagnostic severity, general anxiety symptoms, negative and positive affect, and interference in daily functioning, a series of repeated measures univariate analyses of variance (ANOVAs) were conducted on pre- and posttreatment scores. Effect sizes for ANOVAs are reported as partial eta-squared (η_p^2) for which values of .01, .06, and .14 are considered to reflect small, medium, and large effects, respectively (Cohen, 1973). Analysis of treatment effects on ADIS-IV-L CSRs for principal diagnoses revealed a significant main effect of time ($F_{1,17} = 17.71$, $p = .001$, $\eta_p^2 = 0.51$). To further ascertain pre- to posttreatment effects on individual disorders, separate ANOVAs were run for each of the four anxiety

Table 1
Descriptive Statistics and Effect Size Estimates for Primary Study Variables – Study 1

Measure	N	Pre-Tx		Post-Tx		$F(1,17)$	η_p^2
		Mean	SD	Mean	SD		
ADIS (Co-)Principal Dx CSR	18	5.67	0.69	4.00	2.09	17.71**	0.51
BDI	18	17.50	12.40	11.72	10.53	6.81*	0.29
BAI	18	20.47	8.59	15.18	9.17	10.42**	0.38
PANAS - NA	18	26.50	6.36	22.72	7.99	9.52**	0.36
PANAS - PA	18	28.11	7.08	30.22	6.90	3.53	.172
WSAS	18	3.09	1.50	2.02	1.64	9.59**	0.36

*$p < .05$. **$p < .01$.

disorder categories and depression (DEP) (Table 2). These analyses revealed a significant effect of time for SAD CSRs ($F_{1,7}$=8.61, p=.022, η_p^2=0.55); however, no other disorder was significant (GAD: $F_{1,6}$=3.50, p=.111, η_p^2=0.37; OCD: $F_{1,4}$=4.26, p=.108, η_p^2=0.52; PDA: $F_{1,3}$=9.00, p=.058, η_p^2=0.75; DEP: $F_{1,5}$=6.40, p=.053, η_p^2=0.56), although effect sizes across all disorders were large.

For general symptom measures, a significant main effect of time was found on the BDI ($F_{1,\ 17}$=6.81, p=.018, η_p^2=0.29) and the BAI ($F_{1,\ 17}$=10.42, p=.005, η_p^2=0.38). Finally, analysis of the WSAS also revealed a significant effect of time ($F_{1,\ 17}$=9.59, p=.007, η_p^2=0.36).

Clinical Significance

In order to better determine the clinical significance of the observed effects at posttreatment, we used a conservative adaptation of algorithms reported in other similar trials of CBT for anxiety (e.g., Borkovec, Newman, Pincus, & Lytle, 2002; Ladouceur et al., 2000; Roemer & Orsillo, 2007; Tolin, Maltby, Diefenbach, Hannan, & Worhunsky, 2004) to determine the proportion of individuals meeting criteria for treatment responder status and high end-state functioning. Specifically, individuals were considered to meet responder status if they evidenced a 30% or greater change on at least two measures from the three broad assessment categories (ADIS-IV CSR; WSAS; or diagnosis-specific measures based upon principal diagnosis: BDI, OCI-R, PDSS, PSWQ, SIAS). For example, for an individual with a principal diagnosis of GAD, responder status was determined by looking at change in GAD CSR, WSAS, and PSWQ. Individuals were considered to meet criteria for high end-state functioning if they (a) no longer met diagnostic criteria for their principal diagnosis, as determined by an ADIS-IV CSR of 3 or lower; and (b) fell within normal (subclinical) range on at least one of the remaining broad assessment measures (WSAS or disorder-specific measure). Using this algorithm, 56% of Study 1 participants achieved responder status on their principal diagnosis. Of these treatment responders, half met criteria for high end-state functioning (or 33% of the total sample).

Effects on Comorbidity

To determine the clinical significance of posttreatment effects on comorbid disorders, the same responder and high end-state functioning algorithm was applied to all comorbid disorders, with changes on ADIS CSR and diagnosis-specific measures corresponding to comorbid disorder categories. For example, for an individual with comorbid panic disorder, responder status was determined by looking at change in PDA CSR, WSAS, and PDSS. Using this criteria, 71% of participants achieved responder status on comorbid disorders, with 70% of these attaining high end-state functioning (or 50% of the total sample).

Effects of Treatment on Negative Affect

Analyses of the positive affect (PA) and negative affect (NA) subscales of the PANAS revealed a significant effect of time on NA ($F_{1,17}$=9.52, p=.007, η_p^2=0.36) but not PA ($F_{1,17}$=3.53, p=.077, η_p^2=0.17), although the effect size for PA was also large. Whereas the effect of time on posttreatment NA scores was significant, closer examination of the clinical significance of this change revealed a more modest picture, with just over half of participants (56%) achieving posttreatment scores within the normal range.

Summary

Results from the Study 1 pilot-test of the initial version of the treatment manual provided preliminary support for the efficacy of the UP in the treatment of a range of anxiety and mood disorders including GAD, SAD, PDA, OCD, PTSD, and depression. Treatment with the UP led to an overall reduction in the frequency and severity of both principal and co-occurring disorders from pre- to posttreatment, revealing significant changes in pre- to posttreatment scores across all three of our broad assessment areas, including ADIS-IV CSRs, WSAS, and measures of general anxiety and mood symptoms (BDI, BAI). Further, a separate analysis of treatment effects for specific diagnoses revealed reductions in clinical severity in every diagnosis represented by the sample, including depression. However, only SAD evidenced a significant pre- to posttreatment effect. In addition, a significant change on the NA scale of the PANAS was found, indicating negative

Table 2
ADIS CSRs for Specific Clinical Diagnoses – Study 1

Diagnosis	N	Pre-Tx		Post-Tx		*df*	*F*	η_p^2
		Mean	SD	Mean	SD			
GAD	7	4.86	1.07	3.86	2.12	1, 6	3.50	0.37
SAD	8	5.00	0.93	3.13	2.17	1, 7	8.61 *	0.55
OCD	5	5.20	1.10	3.80	1.92	1, 4	4.26	0.52
PDA	4	5.50	1.00	4.00	1.63	1, 3	9.00	0.75
DEP	6	5.00	0.63	2.33	2.73	1, 5	6.40	0.56

* p<.05.

affect was responsive to treatment, consistent with Brown (2007) above, and these changes were evidenced across diagnoses.

While these initial results were promising, they were nevertheless modest, with just over half of participants achieving responder status, and only one-third of the sample achieving high end-state functioning. In addition, while reductions in clinical severity were evidenced across disorders, diagnoses on average remained at a clinical level (defined as an ADIS CSR at "4" or above) at posttreatment (mean posttreatment CSR = 4.00; *SD* = 2.09). In addition, whereas significant effects were found on a measure of negative affect, the proportion of participants falling within the normal range on NA was still relatively modest. Thus, these preliminary results indicated to us that there was still room for improvement, and highlighted the need for continued treatment refinement and protocol testing. Following an extensive period of further treatment manual development and refinement, the UP was pilot tested in an additional sample of patients. We present these results below in Study 2.

Study 2: Pilot-Test of the Revised UP

Following the initial pilot-test presented in Study 1, and prior to advancing to a more complex randomized controlled trial (RCT), the UP manual underwent several modifications in an effort to improve upon these initial promising results. As suggested by Rounsaville, Carroll, and Onken (2001), this additional treatment manual development phase and further pilot testing allows for thorough testing of the theoretical rationale behind treatment components, and allows for important modifications informed by clinical experience and judgment to be made before moving on to further efficacy and effectiveness testing.

Key Modifications to the UP

The revised version of the UP treatment manual was modified to anchor treatment concepts more explicitly within the three-component, modal model of emotion (see Fairholme, Boisseau, Ellard, Ehrenreich, & Barlow, 2009), and to place a greater emphasis upon increasing patient awareness of the interaction of each of these components within the context of present-moment experience. As the treatment proceeds in the revised manual, the domains of thoughts, feelings, and behaviors are each explored in detail within the context of their contribution to present-moment emotional experiences, focusing specifically on exploring dysfunctional emotion-regulation strategies that the patient has developed over time within each of these domains, and teaching patients more adaptive emotion-regulation skills (for a more detailed description of how emotion-regulation skills are addressed in the UP, we refer the reader to Fairholme et al., in 2009). Treatment sessions from the original protocol were reordered, so that the presentation of core treatment concepts progressed in a more clinically useful and theoretically consistent way.

Specific modifications were as follows:

1. Enhancements were made to the original Session 1 material to expand patients' understanding of the adaptive function of emotions and to promote the development of skills for monitoring their emotional experiences. A description of the ABCs of emotions (antecedent triggers, behavioral responses, and consequences of these responses) was included, as well as specific definitions of the adaptive function of a range of negative emotions, including anger, anxiety, and sadness, and enhanced examples of EDBs triggered by these specific emotions.
2. Emotional awareness training was moved from Session 6 to Session 3 in the revised protocol, emphasizing present-focused, nonjudgmental emotion awareness as an important core skill serving to enhance acquisition of subsequent treatment concepts, including interoceptive exposure. A formal, in-session mindful awareness exercise was also added (adapted from Segal, Williams, & Teasdale, 2002), followed by an emotion-induction exercise using music selected by the patient as emotion provoking.
3. While Sessions 4 and 5 of the initial protocol emphasized antecedent cognitive reappraisal, the revised protocol was modified to reflect more explicitly an emphasis on increasing cognitive flexibility, employing reappraisal strategies not only before but also during and after emotionally laden situations. Additionally, a greater emphasis was placed on teaching patients to recognize how thoughts influence emotions, physical sensations, and behaviors, and vice versa.
4. Session 7 of the revised protocol placed a greater emphasis on using interoceptive exercises not only as a method of exposure to internal cues, but also to build an awareness of how physical sensations interact with and influence thoughts and behaviors. All patients, regardless of diagnosis, were taken through three core interoceptive exposure exercises (breathing through a thin straw, spinning in circles, and hyperventilating).
5. Finally, the revised version of the UP included optional additional "booster sessions," wherein patients solidified acquired emotion-regulation skills through additional emotion-exposure practice. This revised protocol was then pilot-tested in an additional sample of 15 patients seeking treatment at our Center.

Method

Participants

Eighteen patients consented to treatment. Two patients dropped out after the first session of treatment, and 1 patient completed five sessions but was forced to drop out due to transportation difficulties. Therefore, posttreatment data were available for 15 individuals and are reported here. Participants in Study 2 were comparable to Study 1 participants on all demographic variables. Participants in Study 2 were 53.3% female (n=8). The mean age was 29.73 years (SD=7.11) and participants ranged from 18 to 44 years old. The sample for Study 2 was primarily Caucasian (n=12), with 2 participants self-identifying as Asian and 1 participant self-identifying as multiracial. Six individuals were taking psychotropic medications at the time of enrollment and randomization. All individuals were stable on the same dose for at least 3 months prior to enrolling in the study and as part of participation in the study, all agreed to maintain these dosages and medications for the duration of the study. Nine individuals had received prior treatment for anxiety or mood disorders. Overall, the characteristics of Study 2 participants were comparable those included in Study 1.

As in Study 1, any individual with a principal diagnosis of an anxiety disorder (other than a specific phobia) was eligible to participate. Principal diagnoses included: GAD (n=3), SAD (n=5), OCD (n=3), and PDA (n=2). Two individuals had co-principal diagnoses. For these individuals the co-principal diagnoses were GAD and agoraphobia without panic (n=1) and GAD and SAD (n=1). Participants in Study 2 had an average number of 2.2 comorbid diagnoses at pretreatment (SD=1.01; range 1 to 4 diagnoses). Additional or comorbid diagnoses included: GAD (n=3), SAD (n=3), OCD (n=1), PDA (n=2), MDD (n=2), dysthymia (n=1), specific phobia (n=2), hypochondriasis (n=1), and anxiety disorder NOS (n=1).

Measures

Measures and assessment procedures in Study 2 were identical to those in Study 1, with some modifications. Specifically, two additional clinician-administered ratings of symptom severity were added, with clinician-rated assessments conducted by IEs naïve to treatment status or previous assessment ratings (following the same procedure as with the ADIS-IV-L). In addition, the OCI-R was replaced by the Yale-Brown Obsessive Compulsive Inventory (Y-BOCS; Goodman et al., 1989), considered to be the gold standard for assessing obsessive and compulsive symptom severity. Descriptions of these additional assessment measures are provided below.

Structured Interview Guide for the Hamilton Anxiety Rating Scale (SIGH-A; Shear et al., 2001). The SIGH-A was developed to create a structured format for administering the Hamilton Anxiety Rating Scale (HARS; Hamilton, 1959). The SIGH-A includes specific instructions on administration and anchor points for assigning severity ratings. This measure demonstrated good interrater and test-retest reliability. In addition, scores are similar to (although consistently higher than) the HARS.

Structured Interview Guide for the Hamilton Depression Rating Scale (SIGH-D; J. Williams, 1988). Similar to the SIGH-A, the SIGH-D was developed to provide more specific instructions for administration and scoring of the Hamilton Rating Scale for Depression (HRSD; Hamilton, 1960). The SIGH-D also demonstrated good interrater and test-retest reliability and produces scores similar to the HRSD.

Yale-Brown Obsessive Compulsive Scale (Y-BOCS; Goodman et al., 1989). The Y-BOCS is widely regarded as the gold standard for the assessment of obsessive and compulsive symptom severity. For the present study, a 10-item self-report version was used. The self-report version has been shown to have good psychometric properties (Steketee, Frost, & Bogart, 1996).

Treatment

A maximum of eighteen 60-minute individual treatment sessions were allowed in Study 2 (see above for a description of treatment). Patients in Study 2 were seen an average of 17 sessions out of the allowable 18 (range 12–18).

Therapists and Treatment Integrity

Similar to Study 1, therapists for the study were five doctoral students with 1 to 3 years of experience, and one licensed doctoral-level psychologist with 6 years of experience. All therapists provided treatment under the close supervision of licensed senior team members. Treatment adherence was monitored during weekly supervision and manual development meetings.

Results

Effects at Posttreatment Assessment

Descriptive statistics and effect size estimates for the primary study variables are shown in Table 3. As in Study 1, in order to assess the impact of treatment on diagnoses, general anxiety symptoms, negative and positive affect, and interference in daily functioning, a series of repeated measures ANOVAs were conducted on pre- and posttreatment scores. Analysis of treatment effects on ADIS-IV-L CSRs for principal diagnoses revealed a significant main effect of time ($F_{1,\ 14}$=32.17, p=.000, η_p^2=0.70). To further ascertain pre- to posttreatment effects on individual disorders, separate ANOVAs were run for each of the four anxiety disorder categories and DEP (Table 4). These analyses revealed a significant effect of time on ADIS-IV-L CSRs for GAD ($F_{1,\ 5}$=9.49, p=.027, η_p^2=0.66); SAD ($F_{1,\ 8}$=18.18, p=.003, η_p^2=0.69); OCD ($F_{1,\ 3}$=14.47, p=.032, η_p^2=0.83); and PDA ($F_{1,\ 3}$=13.36, p=.035,

Table 3
Descriptive Statistics and Effect Size Estimates for Primary Study Variables – Study 2

Measure	N	Pre-Tx		Post-Tx		*df*	*F*	η_p^2
		Mean	SD	Mean	SD			
ADIS (Co-)Principal Dx CSR	15	5.60	0.83	3.20	1.78	1, 14	32.17***	0.70
SIGH-D	13	13.62	5.64	8.00	6.58	1, 12	9.55**	0.44
SIGH-A	13	14.69	6.74	11.54	7.22	1, 12	4.57	0.28
BDI	14	17.71	9.05	13.00	12.78	1, 13	1.71	0.12
BAI	14	20.86	13.41	12.50	13.48	1, 13	9.28**	0.42
PANAS - NA	14	28.93	7.60	22.29	10.00	1, 13	10.55**	0.45
PANAS - PA	14	29.79	5.60	32.57	4.80	1, 13	2.30	0.15
WSAS	14	3.57	2.24	1.91	1.86	1, 13	7.26*	0.36

*$p<.05$. **$p<.01$. ***$p<.001$.

$\eta_p^2=0.82$). Results for DEP only approached significance ($F_{1,\ 3}=6.94$, $p=.078$, $\eta_p^2=0.70$); however, the effect size was large.

For general symptom measures, a significant main effect of time was found on the SIGH-D ($F_{1,\ 12}=9.55$, $p=.009$, $\eta_p^2=0.44$), but not the SIGH-A ($F_{1,\ 12}=4.57$, $p=.054$, $\eta_p^2=0.28$). Conversely, a significant main effect of time was found on the BAI ($F_{1,\ 13}=9.28$, $p=.009$, $\eta_p^2=0.42$), but not the BDI ($F_{1,\ 13}=1.71$, $p=.213$, $\eta_p^2=0.12$). Finally, analysis of the WSAS also revealed a significant effect of time ($F_{1,\ 13}=7.26$, $p=.018$, $\eta_p^2=0.36$).

Clinical Significance

To determine the clinical significance of effects at posttreatment, we used the same conservative algorithms as in Study 1 above to determine the proportion of individuals meeting criteria for treatment responder status and high end-state functioning. As before, individuals were considered to meet responder status if they evidenced a 30% or greater change on at least two measures from the three broad assessment categories (ADIS-IV CSR; WSAS; or diagnosis-specific measures based upon principal diagnosis: BDI, PDSS, PSWQ, SIAS, or Y-BOCS). Individuals were considered to meet criteria for high end-state functioning if they (a) no longer met diagnostic criteria for their principal diagnosis, as determined by an ADIS-IV CSR of 3 or lower; and (b) fell within normal (subclinical) range on at least one of the remaining broad assessment measures (WSAS or diagnosis-specific measure). Using this algorithm, 73% of Study 2 participants achieved responder status on their principal diagnosis. Of these, 82% met criteria for high end-state functioning (or 60% of the total sample).

Effects on Comorbidity

As in Study 1, to determine the clinical significance of posttreatment effects on comorbid disorders, the same responder and high end-state functioning algorithm was again applied to all comorbid disorders, with changes on ADIS-IV-L CSR and disorder-specific measures corresponding to comorbid disorder categories. Using this criteria, 64% of participants achieved responder status on comorbid disorders, with all of these attaining high end-state functioning (or 64% of the total sample).

Effects of Treatment on Negative Affect

Analyses of the PA and NA subscales of the PANAS revealed a significant effect of time on NA ($F_{1,13}=10.55$, $p=.006$, $\eta_p^2=0.45$) but not PA ($F_{1,13}=2.30$, $p=.153$, $\eta_p^2=0.15$), replicating the results from Study 1. However, a much greater proportion of Study 2 participants evidenced clinically meaningful change in negative affect, with 67% of participants achieving scores within the normal range at posttreatment.

Table 4
ADIS CSRs for Specific Clinical Diagnoses – Study 2

Diagnosis	N	Pre-Tx		Post-Tx		*df*	*F*	η_p^2
		Mean	SD	Mean	SD			
GAD	6	5.17	0.75	3.00	1.79	1, 5	9.49*	0.66
SAD	9	5.22	0.83	3.00	1.73	1, 8	18.18**	0.69
OCD	4	6.00	0.82	2.75	1.26	1, 3	14.47*	0.83
PDA	4	4.75	0.96	3.00	1.83	1, 3	13.36*	0.82
DEP	4	4.50	0.58	2.25	2.06	1, 3	6.94	0.70

*$p<.05$. **$p<.01$.

Table 5
Descriptive Statistics and Effect Size Estimates for Primary Study Variables – Study 2 Follow-Up

Measure	N	Pre-Tx		6-Month Follow Up		*df*	*F*	η_p^2
		Mean	SD	Mean	SD			
ADIS (Co-)Principal Dx CSR	13	5.46	0.78	2.77	1.74	1, 12	32.52***	0.73
SIGH-D	13	13.38	6.16	5.85	3.95	1, 12	16.88**	0.58
SIGH-A	13	13.38	7.03	7.92	4.09	1, 12	6.16*	0.34
BDI	11	16.55	9.33	9.64	11.79	1, 10	2.77	0.22
BAI	11	21.09	13.94	12.45	13.00	1, 10	4.45	0.31
PANAS - NA	11	28.60	8.73	21.20	10.20	1, 10	6.16*	0.41
PANAS - PA	11	32.40	4.03	34.00	7.96	1, 10	0.58	0.06
WSAS	11	3.58	2.53	1.38	1.44	1, 10	7.68*	0.43

*$p<.05$. **$p<.01$. ***$p<.001$.

Treatment Effects at 6-Month Follow-up

Of the 15 treatment completers, 2 participants were unavailable for follow-up assessment, resulting in 13 participants included in the analyses of prolonged treatment effects at 6-month follow-up. In addition, 2 participants did not return self-report questionnaires; therefore, 11 participants are included in the analyses of self-report measures. Descriptive statistics and effect size estimates for the primary study variables at 6-month follow-up are shown in Table 5. Repeated measures ANOVAs were conducted on pre- and 6-month follow-up scores. Analysis of treatment effects on ADIS-IV-L CSRs for principal diagnoses again revealed a significant main effect of time ($F_{1, 12}=32.52$, $p=.000$, $\eta_p^2=0.73$).

For general symptom measures, main effects of time were significant for both the SIGH-D ($F_{1, 12}=16.88$, $p=.001$, $\eta_p^2=0.58$) and the SIGH-A ($F_{1, 12}=6.16$, $p=.029$, $\eta_p^2=0.34$), but were not significant for the BDI ($F_{1, 10}=2.77$, $p=.127$, $\eta_p^2=0.22$), or BAI ($F_{1, 10}=4.45$, $p=.061$, $\eta_p^2=0.31$). Finally, analysis of the WSAS again revealed a significant effect of time ($F_{1, 10}=7.68$, $p=.020$, $\eta_p^2=0.43$).

Clinical Significance at Follow-up

The clinical significance of treatment effects at 6-month follow up were calculated using the same algorithms as before. These analyses revealed 85% of Study 2 participants achieved responder status on their principal diagnosis at 6-months posttreatment. Of these, 82% met criteria for high end-state functioning (or 69% of the total sample). To determine the clinical significance of effects on comorbid disorders at 6-month follow-up, the same responder and high end-state functioning algorithm was again applied to all comorbid diagnoses, with changes on ADIS CSR and diagnosis-specific measures corresponding to comorbid disorder categories. Using this criteria, 80% of participants achieved responder status on comorbid disorders, with 63% of these attaining high end-state functioning (or 50% of the total sample). Table 6 presents

Table 6
Proportion Achieving Responder Status and High End-State Functioning – Study 2

Diagnosis	Post-Treatment			6-Month Follow-Up		
	N	% Treatment Responders	% HES Fx	N	% Treatment Responders	% HES Fx
Principal Only						
All Diagnoses	15	73%	60%	13	85%	69%
Principal or Comorbid						
GAD	6	67%	50%	5	80%	60%
OCD	4	75%	75%	3	100%	100%
PDA	4	50%	50%	4	75%	50%
SAD	9	67%	56%	8	88%	25%
Anx NOS	1	100%	100%	1	100%	100%
Ag w/o Panic	1	0%	0%	1	100%	100%
Depression	4	75%	75%	3	100%	67%
Hypochondriasis	1	100%	100%	1	100%	100%
Specific Phobia	2	50%	50%	2	100%	50%

Note. HES Fx = High end-state functioning. GAD = generalized anxiety disorder. OCD = obsessive compulsive disorder. PDA = panic disorder with agoraphobia. Ag w/o Panic = agoraphobia without panic disorder. SAD = social anxiety disorder.

a breakdown of responder and high end-state functioning rates at posttreatment and 6-month follow-up for principal diagnoses and for each specific diagnosis, regardless of principal or co-morbid status.

Effects of Treatment on Negative Affect

Analyses of the PA and NA subscales of the PANAS at 6-month follow-up again revealed a significant effect of time on NA ($F_{1,10}$=6.16, p=.035, η_p^2=0.41) but not PA ($F_{1,10}$= 0.58, p=.466, η_p^2=0.06). Eighty-two percent of participants had achieved NA scores within the normal range at 6-month follow-up, compared to only 27% at pretreatment.

Summary and Limitations

The results from Study 2 appear to be more robust than from Study 1, with significant improvement evidenced on measures of clinical severity, general symptoms of depression and anxiety, levels of negative affect, and a measure of symptom interference in daily functioning. Importantly, participants in Study 2 evidenced much greater clinically meaningful change, with 73% of participants achieving responder status, and 60% of participants achieving high end-state functioning (as compared to 56% and 33%, respectively, in Study 1). Of note, individuals continued to show improvements beyond termination of the acute treatment phase, with 85% of participants achieving responder status and 69% achieving high end-state functioning at 6-month follow-up. On average, the severity levels of principal diagnoses dropped below diagnostic threshold so that individuals no longer met criteria. This was also found when analyzing specific anxiety disorder categories (GAD, SAD, OCD, and PDA). The effects on comorbid disorders were also promising, with 64% of participants achieving both responder status and high end-state functioning on comorbid disorders. At follow-up, 80% had achieved responder status on comorbid disorders, and over half of these had achieved high end-state functioning. Effects on levels of negative affect were also significant, with 67% of participants scoring within the normal range on the NA scale at posttreatment, as compared to 56% of participants at the end of Study 1. By 6-month follow-up, 82% of participants scored in the normal range on negative affect. This represents a large change from pretreatment scores, which evidenced only 27% within the normal range. These results are particularly intriguing in light of mounting evidence for the paramount role of negative affect as a unifying feature across anxiety and mood disorders, and are consistent with findings that general levels of negative affectivity may be malleable to change (Brown, 2007).

It should be noted that whereas the pre- to posttreatment effect for the BDI reached significance in Study 1, it did not in Study 2. However, treatment effects as measured by the SIGH-D were significant, and were associated with a large effect size. This discrepancy may be related to the presence of an outlier—one participant went from a pretreatment score of 3 to a posttreatment score of 25. This participant was experiencing a high degree of transient acute stress at the time of posttreatment assessment due to a stressful life event. Whereas we cannot rule out the true validity of this patient's posttreatment BDI score, it is nevertheless possible that this score was acutely elevated and may not be a true representation of the patient's progress in treatment. Indeed, at follow-up this same patient obtained a score of 7 on the BDI, no longer met diagnostic criteria on principal diagnosis, and met criteria for high end-state functioning.

Once again, the results presented for both Study 1 and Study 2 are preliminary and should be interpreted with caution because of the small sample size and lack of control condition.

Discussion

In this article, we present preliminary data on outcomes from the UP, a transdiagnostic treatment designed to be applicable across anxiety and mood disorders. Results from a pilot-study of the initial version of the treatment manual, represented in Study 1, provided preliminary support for the efficacy (albeit modest) of the UP in the treatment of a range of anxiety and mood disorders including GAD, SAD, PDA, OCD, PTSD, and depression. After further manual development and modifications to session content, a revised version of the UP was tested in an additional heterogeneous sample of patients, yielding more robust results.

These data suggest that a transdiagnostic treatment distilling common strategies utilized in treating anxiety and mood disorders, enhanced by targeting core affective "higher order" factors, may result in substantial clinical improvement in both principal and comorbid disorders. If this is the case, clinicians are afforded a much more parsimonious approach to treatment planning (Mansell et al., 2009) that eliminates the need for multiple diagnosis-specific treatment manuals and more cumbersome treatment planning. This approach to the treatment of emotional disorders, if verified as successful, may prove valuable in the dissemination of evidence-based treatments, removing some of the traditional barriers to their implementation, such as the significant time and cost required for adequate training in multiple treatment manuals (Addis, Wade, & Hatgis, 1999). Moreover, as clinicians are often faced with the task of treating patients with complex clinical presentations, the use of a single protocol eliminates the need to use multiple protocols to tackle several problems at once, which has been shown to result in poorer treatment outcome (Craske et al., 2007).

Second, the results of these studies lend some indirect support to a more dimensional conceptualization of psychopathology. In the present study, targeting core affective factors rather than diagnosis-specific symptoms resulted in clinically significant changes across a range of anxiety and mood disorders, including both principal and co-occurring diagnoses. As comorbidity in clinical samples tends to be the rule rather than the exception (Brown et al., 2001), the arbitrary splitting brought about through categorical methods of diagnosis may not accurately capture or address the dynamic and interacting nature of these disorders, or the true holistic experience of these patients. Moving away from targeting disorder-specific symptoms and towards factors existing along the full "neurotic spectrum" may prove both more parsimonious and more experientially accurate (Brown & Barlow, 2005; Brown & Barlow, in 2009).

Finally, our results speak to the necessity of testing and refining treatments to improve their feasibility, acceptability, and clinical utility. Refining our protocol based on the results from Study 1 and the clinical experience accrued from administering the protocol resulted in a revised protocol that was both more internally consistent and, seemingly, efficacious. Currently in its final stages of development and testing, the UP (version 3.0) has undergone additional changes informed by the outcomes data presented above, and direct use of the protocol in clinical practice. The principal changes in UP version 3.0 include additional techniques for enhancing motivation to engage in treatment, drawing from the work of Miller, Rollnick, Arkowitz and Westra (Arkowitz & Westra, 2004; Arkowitz, Westra, Miller, & Rollnick 2008; Miller & Rollnick, 1991, 2002); a greater focus on the role of positive emotion, both as a trigger for maladaptive emotion avoidance and as a target for emotion exposures; and expanded discussion of several key principles.

In addition to these changes, the latest version of the UP includes a shift from session-by-session content to a modular approach to treatment. Each of the core treatment concepts (i.e., present-focused emotional awareness, cognitive flexibility, countering emotional avoidance and emotion-driven behaviors, interoceptive and situation-based emotion exposures) are encapsulated within individual modules, intended to be delivered within a range of one to three sessions. Consistent with the defining principles of modularity as described by Chorpita, Viesselman, and Hamilton (2005), the modularized version of the UP is expected to provide clinicians with greater flexibility in the presentation to patients of core treatment concepts and skills, thus enhancing opportunities for skill acquisition and promoting more individualized patient care. In addition, the modular approach opens the possibility for a more "prescriptive" approach to treatment, wherein deficits in core skills corresponding to specific modules can be assessed in order to determine which of the modules ought to be applied or the amount of time that ought to be spent on each particular module. This prescriptive approach offers a number of possible advantages over using multiple manualized protocols, including greater efficiency in the administration of treatment procedures, greater cost-effectiveness, improved transportability across treatment contexts, and potentially improved treatment efficacy. However, the efficacy of a prescriptive approach is yet to be determined.

We are currently in the process of collecting data on the most recent version of the UP in a National Institute of Mental Health supported RCT. In order to determine efficacy of the UP against a control condition, modularity in the current version is being limited to allowing flexibility in the number of allowable sessions for each core concept within a predetermined range. As such, the version of the protocol currently being tested does not represent a radical departure from the version used in Study 2 reported here. Future studies are needed to examine the effectiveness and transportability of the transdiagnostic approach, as well as the applicability of the UP to other disorders in which emotion plays a key role, such as somatoform and dissociative disorders. Further, follow-up data are needed to determine long-term clinical utility of the UP. In addition, dismantling studies are needed to evaluate whether all of the core skills presented in the UP are necessary for treatment gains. Finally, it remains an empirical question whether taking a modular approach could lead to a more prescriptive approach to treatment, wherein specific decision rules lead to more individualized delivery and "dosing" of treatment concepts. In anticipation of these important future investigations, these initial findings lend encouraging preliminary support for the UP as an efficacious, transdiagnostic treatment for emotional disorders.

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Received: March 15, 2009
Accepted: June 13, 2009
Available online 29 January 2010